serotype of the patient's illness. By food history, honey exposure was found to be a significant risk factor for type B infant botulism. Worldwide, 35 percent of babies with infant botulism were fed honey (but it is important to note that 65 percent were not). In a separate survey, *C botulinum* was found in retail and producer honey samples from nine widely separated states, and these samples were not associated with cases of infant botulism. Clearly, contamination of honey with spores occurs before the honey reaches the home. Using current laboratory methods, about 10 percent of honey has been found to contain *C botulinum.*

There is insufficient information with which to decide whether or not honey may play a role in SIDS. In none of the ten rapidly fatal cases of infant botulism diagnosed by examination of autopsy specimens had the infants been fed honey. Of the three infants that died from infant botulism after admission to hospital, two had been fed honey and one had not.

Honey is the one identified, avoidable source of *C botulinum* spores for susceptible infants. Because honey is not essential for infant nourishment, it may be omitted from infants' diet, and should be. As 65 percent of cases had no honey exposure, elimination of honey from infant foods will not eradicate all infant botulism. However, omission of honey from infants' diet is the first step—and at present the only practical preventive measure for infant botulism that parents and health care providers can practice. The safety of honey for older persons with normal intestinal physiology remains unquestioned.

STEPHEN S. ARNON, MD

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Haemophilus influenzae: Secondary Spread

REPORTS OF THE SPREAD of serious infections due to *Haemophilus influenzae* type B within families and at day-care centers suggest that young children are at increased risk of contracting serious disease due to this organism within one month of illness in a close contact situation. It has been well established that the risk of secondary cases among household contacts of patients with meningococcemia or meningococcal meningitis is approximately three per 1,000. Recently, similar estimates of the frequency of secondary illness in household and day-care center contacts of children with serious disease due to *H influenzae* have become available.

Filice and co-workers have estimated the risk of serious secondary illness to be 0.4 percent in all household contacts of patients with *H influenzae* meningitis. They report a secondary attack rate for contacts two years of age and younger of 4.9 percent. A larger prospective study conducted by the Center for Disease Control involved a one-month follow-up of all household contacts of 1,147 cases of *H influenzae* meningitis. Nine cases of serious disease due to *H influenzae* were detected, all in children less than 6 years of age. The age-specific risk of secondary disease was as follows: younger than 1 year, 5.0 percent; 1 year, 1.4 percent; 2 to 3 years, 1.5 percent; 4 to 5 years, 0.1 percent.

Ward and co-workers have reported similar findings in a day-care facility. During an eight-day period, serious disease due to *H influenzae* developed in three of 75 children, 5 months to 6 years of age. All three patients were less than 1 year of age. The attack rate at the day-care center was 4 percent for all children, and 25 percent for infants. Approximately 5 percent of the children at the day-care center were found to be nasopharyngeal carriers of *H influenzae.* All staff were culture negative. Three of five siblings and two of ten parents of infected children had positive nasopharyngeal cultures.

These studies suggest that young children are at increased risk of contracting serious diseases due to *H influenzae* within the month after onset of illness in close contacts. Therefore, prophylaxis of infants and children less than 6 years of age should be considered. Rifampin, cotrimoxazole and ampicillin have been suggested as prophylactic agents. Only limited data are available on the efficacy of these agents, and to date only rifampin appears to be effective in eradicating nasopharyngeal carriage. The dosage of rifampin is 10 mg per kg of body weight per day (not to exceed 600 mg per day) for four days. Adverse effects are unusual; they include gastrointestinal disturbances, headache, drowsiness, ataxia and hypersensitivity reactions. Hepatic, hematologic and renal abnormalities are usually transient. More

studies are needed before recommendations regarding prophylaxis can be made. Prophylaxis of contacts more than 6 years of age is probably not justified.

LARRY J. BARAFF, MD

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Splenic Trauma in Children

RUPTURE OF THE SPLEEN is the most common intra-abdominal injury occurring in children following blunt trauma. Emergency laparotomy and splenectomy are customarily carried out. However, an increasing awareness of the syndrome of overwhelming postsplenectomy infection (OPSI) has caused many pediatricians and surgeons to reevaluate this form of treatment.

While the incidence of sepsis following splenectomy secondary to trauma is low, the associated mortality is 58 times greater than that reported for similar infections in the general population. Classically, these infections occur abruptly with a florid bacteremia and a high incidence of disseminated intravascular coagulation. Death, frequently occurring within hours of onset, is seen in 80 percent of cases. *Diplococcus pneumoniae* is the causative organism in most instances, followed by *Haemophilus influenzae* and *Neisseria meningitidis*. Children less than 5 years of age are usually affected and, in most cases, OPSI is seen within two years following splenectomy. Several patients, however, have been adolescents at the time of splenectomy and infections have occurred as late as 15 years following splenectomy.

Due to the high mortality associated with OPSI, penicillin prophylaxis for up to two years following splenectomy has been advocated, but efficacy of this regimen has not been proved. Pneumococcal vaccine is now available for clinical use; however, this vaccine is only effective against 80 percent of pneumococcal infections and is not recommended for children less than 2 years old. There are reported instances of pneumococcal sepsis in postsplenectomized persons who have received pneumococcal vaccine that includes the type of organism responsible for the infection.

Alternative methods of management have recently been proposed for children with traumatic rupture of the spleen because of the possible risk of OPSI. New surgical techniques have been developed that make it possible for the injured spleen to be repaired and preserved. Several clinical reports have been published showing the feasibility and safety of this form of treatment. Another approach is nonoperative observation after confirming the diagnosis of an isolated splenic rupture by either splenic scan or arteriogram. In such instances, the children are admitted to an intensive care facility where their conditions can be carefully monitored. Surgical operation is not done if a patient's hematocrit stabilizes within 48 hours and the need for transfusion does not exceed a third of the patient's blood volume. Twenty patients have been treated in this manner at the Children's Hospital Medical Center in Boston. Splenectomy was not required in any patient and there were no late sequelae.

An interesting article by Pearson and co-workers suggests that splenosis following traumatic splenectomy is more common than previously thought and may protect against OPSI. In 13 of 22 children there was splenic activity one to eight years following traumatic splenectomy. Splenic scans done on five children showed significant extrahepatic uptake of radionuclide. The protective effect of splenosis is open to question, however, because autopsies in several fatal cases of OPSI following traumatic splenectomy have shown significant splenosis or accessory spleens. This finding supports the experimental evidence that the quantity of splenic tissue and the vascular supply to this tissue are important factors in the prevention of pneumococcal sepsis. Several studies also indicate certain specific splenic functions act independently of others. Consequently, the ability of splenic tissue to remove degenerate red cells does not necessarily correlate with its ability to prevent pneumococcal sepsis.

Based on the above discussion, the following recommendations can be made. A child in whom a splenectomy has been done should be given a pneumococcal vaccine. Penicillin prophylaxis should probably be instituted as well and continued for at least two years following splenectomy. While the efficacy of penicillin prophylaxis has not been well documented, many physicians recommend lifelong treatment with penicillin in children after splenectomy. When an emergency laparotomy is carried out in a child and an isolated splenic injury found, splenic repair should